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У збірнику представлені матеріали 101 – ї підсумкової наукової конференції професорсько-викладацького персоналу вищого державного навчального закладу України «Буковинський державний медичний університет» (м.Чернівці, 10, 12, 17 лютого 2020 р.) із стилістикою та орфографією у авторській редакції. Публікації присвячені актуальним проблемам фундаментальної, теоретичної та клінічної медицини.

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THE CONTENTS OF CYTOKERATIN 18, ADIPONECTIN AND LEPTIN IN PATIENTS WITH COMORBID COURSE OF NONALCOHOLIC STEATOHEPATITIS AND CORONARY HEART DESEASE

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One of the most informative markers of the inflammation and liver fibrosis is the presence of the cytokeratin 18 (CK 18). CK 18 is a fragment of intermediate phylums of cells cytoskeleton, which is cut by the effector caspase 3 because of the process of apoptosis of hepatocytes. This peptide is detected in the blood even before the morphological signs of apoptosis occur. It allows to use CK-18 as a non-invasive biomarker of NASH.

The objectives of the study was to determine the level of cytokeratin 18, adiponectin and leptin in patients' plasma of with NASH and comorbid coronary heart disease (CHD) and their association with the degree of cytolysis, disorders of carbohydrate and lipid metabolism.

We examined 60 patients with NASH, which were divided into 2 groups: group 1 - 30 patients with NASH on the background of obesity of I-II degree, group 2 - 30 patients with NASH and comorbid CHD (the I and II functional classes of the stable angina pectoris) and obesity of I-II degrees. The average age of patients was 55.13 ± 4.34 years. The control group consisted of 20 practically healthy persons (PHP). Serum CK18, adiponectin and leptin levels were determined by the immunoassay analysis.

Our research showed that NASH is closely associated with disorders of adipocytokine homeostasis. In patients with NASH and comorbid CHD and obesity of I-II degree there is an increase level of CK 18, proinflammatory adipokine leptin and a decrease in the level of adiponectin.

Thus, the significant increase of liver aminotransferase activity and the level of CK 18 fragment in patients with NASH, their close positive correlation relationship suggest that the leading pathophysiological mechanism of progression of NAFLD is the necrosis and apoptosis of hepatocytes. The determination of serum fragments of CK 18 can be used as a non-invasive test for diagnostic of NASH and liver steatosis.

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CURRENT VIEWS ON PATHOGENESIS OF THE ADVERSE PARTICULAR INFLUENCE OF ULTRADISPERSE PARTICLES ON THE CARDIOVASCULAR SYSTEM

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Evidence on the health effects of ultrafine particles (UFP) is still limited as they are usually not monitored routinely. Organs that might be affected by fine and ultrafine particles are not only the lungs but also the cardiovascular system and other organs such as the brain. Although epidemiological studies by nature can never prove hypothesized pathways, the large number of studies forms a sound basis for evidence and the main pathways described are now widely accepted.

Aim: To analyze the current views on pathogenesis of the harmful effects of UFP on the cardiovascular system. Research methods: informational-analytical, content-analysis.

The analysis of the results showed that despite the fact that the precise mechanisms by which UFP affect the cardiovascular system are under study, several probable pathways have already been described. Thus, three generalized intermediate mechanisms through which UFP can affect the cardiovascular system have been proposed, but none of them can work separately, and the complex combination and interaction of the mechanisms is not yet fully understood.

These three main mechanisms are: Systemic inflammation: Numerous experimental and epidemiological studies have shown that inhalation of UFP causes increased accumulation and